Claim 1 (Cancelled). A method for stimulating angiogenesis within a targeted collection of viable cells in-Situ, said method comprising the steps of:

identifying a collection of cells comprising viable cells in-situ as a target for stimulation of angiogenesis;

providing means for effecting an introduction of at least one member selected from the group consisting of the PR-39 oligopeptide collective to the cytoplasm of said targeted collection of cells;

introducing at least one member of the PR-39 oligopeptide collective to the cytoplasm of said targeted collection of cells using said effecting means;

allowing said introduced PR-39 oligopeptide collective member to interact with such proteasomes as are present within the cytoplasm of said targeted collection of cells whereby

(a) the $\alpha 7$ subunit of at least some of the proteasomes interact with said PR-39 oligopeptide collective member, and

(b) the proteolytic degradation of at least one identifiable peptide mediated by said proteasomes with an interacting $\alpha 7$ subunit becomes markedly inhibited while the proteolytic degradation mediated by said proteasomes with an interacting $\alpha 7$ subunit against other individual peptides remains unaltered, and

(c) the markedly inhibited proteolytic degradation activity of said

proteasomes with said interacting α7 subunit results in a stimulation of angiogenesis in-situ.

Claim 2 (Cancelled). A method for altering proteasome-mediated degradation of peptides in-situ within a collection of viable cells, said method comprising the steps of:

identifying a collection of cells comprising viable cells in-situ as a target;

providing means for effecting an introduction of at least one member selected from the group consisting of the PR-39 oligopeptide collective to the cytoplasm of said targeted collection of cells;

introducing at least one member of the PR-39 oligopeptide collective to the cytoplasm of said targeted collection of cells using effecting means;

allowing said introduced PR-39 oligopeptide collective member to interact with such proteasomes as are present within the cytoplasm of said targeted collection of cells whereby

(a) the α7 subunit of at least some of the proteasomes interacts with the PR-39 oligopeptide collective member, and

(b) the proteolytic degradation of at least one identifiable peptide mediated by said proteasomes with an interacting α7 subunit becomes markedly inhibited while the proteolytic degradation mediated by said

interacting proteasomes with an interacting $\alpha 7$ subunit against other individual peptides remains unaltered, and

(c) the markedly inhibited proteolytic degradation of the proteasomes with said interacting $\alpha 7$ subunit results in an increased expression of said identifiable peptide in-situ within the targeted collection of cells.

Claim 3 (Cancelled). The method as recited in claim 1 or 2 wherein said collection of viable cells includes at least one type of cell selected from the group consisting of endothelial cells, myocytes and myoblasts, fibrocytes and fibroblasts, epithelial cells, osteocytes and osteoblasts, neuronal cells and glial cells, erythrocytes, leukocytes, and progenitor cells of all types.

Claim 4 (Cancelled). The method as recited in claim 1 or 2 wherein said collection of cells comprises at least one tissue selected from the group consisting of myocardium, skeletal muscle, smooth muscle, an artery, a vein, lung, brain, kidney, spleen, liver, gastrointestinal tissue, nerve tissue, limbs, and extremities.

Claim 5 (Cancelled). The method as recited in claim 1 or 2 wherein the means for an introduction of a PR-39 oligopeptide collective member include one selected from the group consisting of catheter-based means, injection-

based means, infusion-based means, localized intravascular means, liposome-based means, receptor-specific peptide means, and slow releasing means for peptide secretion in living cells and sequestered organisms.

Claim 6 (Cancelled). The method as recited in claim 1 or 2 wherein the means for an introduction of a PR-39 oligopeptide collective member includes DNA sequences coding for at least one PR-39 oligopeptide collective member in an expression vector for transfection and subsequent expression of the PR-39 oligopeptide collective member within said cells.

Claim 7 (Cancelled). The method as recited in claim 1 or 2 wherein said method is practiced under in-vivo conditions.

Claim 8 (Cancellled). The method as recited in claim 1 or 2 wherein said method is practiced under in-vitro conditions.

Claim 9 (Cancelled). The method as recited in claim 1 or 2 wherein degradation of IκBα is inhibited.

Claim 10 (Cancelled). The method as recited in claim 1 or 2 wherein degradation of HIF- 1α is inhibited.

Claim 11 (Currently amended): A PR-39 derived oligopeptide family whose members individually are ~~pharmacologically active and functionally specific~~ operative and functional to cause a selective inhibition of proteasome-mediated degradation in-situ after introduction intracellularly to a viable cell, each member of said PR-39 derived oligopeptide family being

a pharmacologically active oligopeptide which is less than 26 amino acid residues in length;

a pharmacologically active oligopeptide whose N-terminal amino acid residue sequence begins with Arg-Arg-Arg;

a pharmacologically active oligopeptide which is an analog of the amino acid sequence of native PR-39 peptide;

a pharmacologically active oligopeptide ~~able~~ operative to selectively alter the proteolytic degradation activity of proteasomes in-situ;

a pharmacologically active oligopeptide ~~able~~ operative to interact in-situ with at least the α7 subunit of such proteasomes as are present within the cytoplasm of the cell; and

a pharmacologically active oligopeptide ~~able~~ operative selectively to alter the proteolytic degradation activity of said proteasomes having an interacting α7 subunit such that the proteolytic degradation mediated by said proteasomes against at least one peptide selected from the group consisting of NFκB_inhibitor IκBα and hypoxia-inducing factor (HIF)-1α becomes

selectively inhibited without substantially altering the proteolytic degradation of other peptides mediated by said proteasomes.

Claim 12 (Previously Presented): The PR-39 derived oligopeptide family as recited in claim 11 or 15 whose membership includes a peptide comprised of 15 amino acid residues whose sequence is Arg-Arg-Arg-Pro-Arg-Pro-Pro-Tyr-Leu-Pro-Arg-Pro-Arg-Pro-Pro (SEQ ID NO: 3).

Claim 13 (Previously Presented): The PR-39 derived oligopeptide family as recited in claim 11 or 15 whose membership includes a peptide comprised of 11 amino acid residues whose sequence is Arg-Arg-Arg-Pro-Arg-Pro-Pro-Tyr-Leu-Pro-Arg (SEQ ID NO: 4).

Claim 14 (Previously Presented): The PR-39 derived oligopeptide family as recited in claim 11 or 15 whose membership includes a peptide comprised of 8 amino acid residues whose sequence is Arg-Arg-Arg-Pro-Arg-Pro-Pro-Tyr (SEQ ID NO: 5).

Claim 15 (Currently Amended): A PR-39 derived oligopeptide family whose members individually are ~~pharmacologically active and functionally specific~~ operative and functional to cause a selective inhibition of protease-

mediated degradation in-situ after introduction intracellularly to a viable cell, each member of said PR-39 oligopeptide family being:

a pharmacologically active oligopeptide which is less than 20 amino acid residues in length;

a pharmacologically active oligopeptide whose N-terminal amino acid residue sequence begins with Arg-Arg-Arg;

a pharmacologically active oligopeptide which is an analog of the amino acid sequence of native PR-39 peptide;

a pharmacologically active oligopeptide ~~able~~ operative to selectively alter the proteolytic degradation activity of proteasomes in-situ;

a pharmacologically active oligopeptide ~~able~~ operative to interact in-situ with at least the $\alpha 7$ subunit of such proteasomes as are present within the cytoplasm of the cell; and

a pharmacologically active oligopeptide ~~able~~ operative selectively to alter the proteolytic degradation activity of said proteasomes having an interacting $\alpha 7$ subunit such that the proteolytic degradation mediated by said proteasomes against at least one peptide selected from the group consisting of NFκB_inhibitor IκBα and hypoxia-inducing factor (HIF)-1α becomes selectively inhibited without substantially altering the proteolytic degradation of other peptides mediated by said proteasomes.